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DOI: https://doi.org/10.1136/vr.100657

Originally published at:
Golini, Lorenzo; Morgan, Joe P; Glaus, Tony M; Steffen, Frank (2012). Successfull medical treatment of Erysipelothrix rhusiopathiae-induced lumbosacral discospondylitis in a dog. Veterinary Record, 170(21):543b.
DOI: https://doi.org/10.1136/vr.100657

Veterinary Record

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Journal:	*Veterinary Record*
Manuscript ID:	vetrec-2012-100657.R2
Article Type:	Short communication
Date Submitted by the Author:	17-Feb-2012
Complete List of Authors:	Golini, Lorenzo; VetSuisse Faculty, University of Zurich, Department of Small Animal, Neurology Service Morgan, Joe; Vetsuisse Faculty University of Zurich, Department of Small Animals, Section of Diagnostic Imaging Glaus, Tony; University of Zurich, Steffen, Frank; VetSuisse Faculty - University of Zurich, Department of Small Animals, Neurology Service

Successful medical treatment of *Erysipelothrix rhusiopathiae*-induced lumbosacral discospondylitis in a dog

Golini, L. DVM MSc(Behavior)[1] , Morgan, J.P. Prof. Dr. Med.Vet. DACVR[2], Glaus, T Jr. Prof. Dr Med Vet DACVIM (Internal Medicine) DECVIM (Cardiology)[3], Steffen F PD Dr Med Vet DECVN[1],

1. Neurology Service, Department of Small Animals, VetSuisse Faculty, University of Zurich, Winterthurerstrasse 260, 8050 Zurich, Switzerland

2. Section of Diagnostic Imaging, VetSuisse Faculty, University of Zurich, Winterthurerstrasse 260, 8050 Zurich, Switzerland.

3. Cardiology Service, Clinic for Small Animal Internal Medicine, VetSuisse Faculty, University of Zurich, Winterthurerstrasse 260, 8050 Zurich, Switzerland

Address correspondence to: Dr. L. Golini (lgolini@vetclinics.uzh.ch)

The lumbosacral disk is a common site for the settlement of local or systemic bacterial infections, leading to lumbosacral discospondylitis (Burkert and others 2005; Gilmore 1987). More commonly isolated organisms include *Staphylococcus spp*., *Brucella canis*, *Streptococcus spp*. and *Escherichia coli,* additional less common organisms include *Pasteurella multocida*, *Actinomyces viscosus*, *Nocardia spp*., *Mycobacterium avium*, *Proteus spp*. and *Corynebacterium spp*. (Betbeze and McLaughlin 2002; Burkert and others 2005). In human medicine, pyogenic vertebral osteomyelitis is mainly due to *Streptococcus aureus*, followed by *E. coli*, Coagulase-negative staphylococci and *Propionibacterium acnes* (Zimmerli 2010). *Erysipelothrix rhusiopathiae* is rarely recognized as zoonotic agent of osteomyelitis in immunodeficient patients but it is easily treatable. Whereas this organism is a very important pathogen in pigs, where acute disease is characterized by sudden death or general signs of septicaemia, the sub acute form is characterized by classical diamond-skin light pink to reddish lesions or a chronic form could result with signs of local arthritis or proliferative pathological changes in the heart (Wang and others 2010). To the authors' knowledge, *E. rhusiopathiae* has only been described in 3 fatal canine cases, of which 2 dogs had discospondylitis as part of the disease (Burkert and others 2005; Houlton and Jefferies 1989; Seelig and others 2010). The present case report illustrates the clinical and radiological features; diagnostic procedure, clinical cure and long term clinical follow up of a dog diagnosed with *Erysipelothrix rhusiopathiae*-induced lumbosacral diskospondylitis.

The dog, a 13year old male Labrador retriever, was presented to the Neurology Service of the Veterinary Teaching Hospital Faculty of Veterinary Medicine University of Zurich for a second opinion about a long term problem in failure to jump and walk normally. The dog had been living in Ibiza, Spain, since it was born where it was regularly vaccinated, yearly de-wormed and during the warm season was wearing a deltametrine based collar to reduce the risk of phlebotomus bites. Since two months before presentation the dog had shown apathetic behaviour, progressive worsening of difficulties to walk normally and jump into the car, with occasional tenesmus and uncontrollable pain during petting the lumbosacral area. At clinical examination, the dog was apathetic, febrile (40,3°C) and panting, the mucous membranes were mildly reddish and the heart rate was 150 beats per minutes. The heart auscultation was mildly arrhythmic without murmurs. The dog had no neurological deficits, however, he showed pain on palpation of the caudal abdomen and in the lumbosacral area as well as pain upon extension of the hips (lordosis test) and trans-rectal palpation of the ventral aspect of the sacrum. On rectal palpation, the prostate was moderately enlarged but not painful. Lumbosacral disease was clinically suspected and an infectious cause was presumed; degenerative, neuropathic or neoplastic disease involving the cauda equina or the bone and joints structures of this anatomical area were considered less likely.

Radiographs of thorax, caudal abdomen, lumbosacral area and hips of the dog were performed under sedation (should this be anaesthesia from what follows below?). Thorax, abdomen and hips were unremarkable and the vertebrae between C6 and L6 showed no obvious pathological changes. A heavy pattern of spondylosis deformans was present around L6-7 and L7-S1 ventrally and laterally. The endplates of the vertebrae L7-S1 were sclerotic with associated intervertebral disc space collapse. Lytic/destructive lesions in the mid portion of the endplates were also observed (Fig.1 and Fig.2). The radiological diagnosis supported the clinical suspicion of discospondylitis at the lumbosacral joint superimposed to chronic spondylosis deformans.

With the dog still under anaesthesia, a fluoroscopically guided fine needle aspiration of the L7-S1 disk was performed. A 20G 90 mm long spinal needle was aseptically inserted perpendicularly through the surgically prepared skin into the disk (Fig. 1), and gentle aspiration was performed with a sterile 10 cc syringe. The aspirated material was gently deposited on a sterile transport swab, suitable for both aerobes and anaerobes (COPAN Innovation, Brescia, Italy). A second sample was obtained by inserting the needle on a different path from the first one and flushing the disk with 1 ml of sterile and pyrogen – free irrigation solution (NaCl 0.9%, B. Braun Melsungen AG, Germany). The re-aspirated fluid was transported to the laboratory by means of anothersterile transport swab similar to previous one. Finally, ultrasound examination of the heart was performed and did not reveal any abnormalities, specifically, all cardiac valves were free of any suspicion of endocarditis.

Routine haematological examination showed severe leukocytosis (65.000 WBC/ul; reference range: 4.700 – 11.300 cells/ul) with a left shift (6000/ul bands; reference range: 0-800 cells/ul) and monocytosis (1500/ul; reference range: 200 – 920 cells/ul). Urine analysis obtained by cystocentesis did not reveal pyuria or bacteria, but urine was cultured nevertheless. Ultrasound evaluation of the prostate gland and of the testicles did not reveal any abnormalities other than expected with older age, and a fine needle aspiration of the prostatic tissue was cytologically unremarkable. Blood cultures for aerobic and anaerobic bacteria were taken, sampling 10 ml of whole blood hourly three times and putting the blood into a commercially available transportation/culture medium (BC0102M, Oxoid SIGNAL blood culture system medium; Oxoid Limited, Basingstoke, UK).

Recovery from anaesthesia was uneventful and the dog was administered first generation intravenous cefazolin (Kefzol, TEVA Pharma AG, Basel, Switzerland) at

22 mg/Kg/TID. General physical condition, gait abnormalities and pain improved significantly by the following day. After 24 hours of intravenous antibiotic treatment, the dog was discharged with long-term oral antibiotic treatment at the same dosage (Cefaseptin forte, Vétoquinol AG, Switzerland), to be modified based on microbiological culture and sensitivity results.

No etiological agent growth was obtained from either urine culture or blood cultures while from the disk material a small Gram-positive bacillus was cultured from both the FNA of the disk and the disk wash. An amplification of 1600 base pairs with conventional PCR for the 16S rDNA of the bacterium was performed, according to the protocol developed by Medlin et coll (Medlin and others 1988), and using as forward primer the sequence 5'-CAG AGT TTG ATC CTG GCT CAG-3' and as reverse primer 5'-TAC GG(CT) TAC CTT GTT ACG ACT T-3'. Afterwards, the amplified gene was sequenced and compared with an online gene bank (http://blast.ncbi.nlm.nih.gov; accessed on March 2010). The sequenced gene showed a perfect homology with the available sequences of *Erysipelothrix rhusiopathiae*. The same procedure was subsequently performed on the blood sampled previously for culture, leading again to the identification of DNA of *Erysipelothrix rhusiopathiae*. The antibiogram showed that it was sensitive to cefazolin and therefore this treatment was continued for 3 months. Regular telephone follow-up confirmed the resolution of all clinical signs at 20 days after discharge, and after 6 weeks the dog was jumping and running normally. After 3 months, the referring veterinarian stopped the treatment and at the time of writing, 9 months after diagnosis, the dog has not been showing any recurring clinical signs.

Discospondylitis is an infection of the intervertebral disc that spreads through both endplates and proceeds slowly into the two contiguous vertebrae (Gilmore 1987).

Although any intervertebral disk space may be affected, the lower cervical, the midthoracic, the thoracolumbar and the lumbosacral disks are more often involved in dogs (Burkert and others 2005; Gilmore 1987; Kinzel and others 2005; Kornegay and Barber 1980). Clinical signs range widely from pain alone to non-ambulatory para- or tetraparesis, either flaccid or spastic, depending upon the localization of the inflammatory and compressive process (Betbeze and McLaughlin 2002; Burkert and others 2005; Kornegay and Barber 1980). Male dogs are twice as likely to be affected as females and the risk increases with age (Burkert and others 2005). Additional predisposing factors are previous surgery, treatment with immunosuppressive drugs, or systemic or local infections, most commonly located in the lower urinary tract (Burkert and others 2005). In intact male dogs, a common infectious route to affect the lumbosacral disc is through the lymphatic vessels that drain the prostate gland with subsequent arterial spread. Primary hematogenous spread is also a recognized route of infection (Betbeze and McLaughlin 2002; Burkert and others 2005; Gilmore 1987) Rarely, penetrating wounds, migrating foreign bodies, like foxtail or other plant thorns, or even more seldom sterile procedures, as epidural punctures, can convey bacteria into the affected site (Betbeze and McLaughlin 2002; Corlazzoli and Pizzirani 1998; MacFarlane and Iff 2011).

For the aforementioned reason urine culture should be performed systematically in all dogs with lumbosacral discospondylitis even in absence of pyuria, even though urine culture are negative in up to 40% of the cases (Burkert and others 2005; Gilmore 1987; Kornegay and Barber 1980). The combination of urine and blood cultures yield a successful etiological diagnosis in more than 70% of the cases (Betbeze and McLaughlin 2002; Burkert and others 2005; Gilmore 1987; Kinzel and others 2005). Pre-treatment of affected animals with antibiotics could lead to false negative culture results. Finally, as also performed in the case reported here, a direct biopsy of the inflamed tissue either by fluoroscopic guidance or computer tomography and by direct surgical sampling in order to isolate and identify the responsible bacteria is an accurate diagnostic procedure in 76% to 100% of the cases (Gilmore 1987; Kinzel and others 2005; Kornegay and Barber 1980; Vignoli and others 2004; Zimmerli 2010).

A multitude of bacteria are commonly isolated from dogs with discospondilyitis, however, *Erysipelothrix rhusiopathiae* has only been described in two clinical reports with discospondylitis in which dogs showed widespread joint inflammation, endocarditis and discospondylitis at multiple sites (Burkert and others 2005; Houlton and Jefferies 1989; Seelig and others 2010). Furthermore, besides polyarthritis and aortic valve endocarditis, discospondylitis was also found in 8 of 9 Beagle dogs experimentally infected with *Erysipelothrix rhusiopathiae* (Houlton and Jefferies 1989; Schütt and others 1978).

E. rhusiopathiae is a facultative, non-spore-forming, non-acid-fast, small, Gram-positive bacillus. Its distribution is worldwide and it can survive for long periods in the soil as well as in marine environment(Wang and others 2010). Interestingly, *E. rhusiopathiae* is known to be a natural host of the slim of some fishes without causing any apparent disease (Wang and others 2010). As for other cases, the route of infection in our patient was not determined.

To the author's knowledge, this is the first clinical report describing the successful treatment of lumbosacral discospondylitis due to *E. rhusiopathiae.* The initial antibiotic choice was a first generation cephalosporin, which is suggested by several authors as drug of choice when the causing organism is unknown, and which then was proven to be effective in our case based on the sensitivity results and clinical course

(Burkert and others 2005; Gilmore 1987; Kornegay and Barber 1980). The duration of antibiotic treatment in discospondylitis does not have a fixed rule and has ranged from 2 to 130 weeks in various reports (Betbeze and McLaughlin 2002; Burkert and others 2005; Gilmore 1987). It is mainly guided by radiological evidence of bone healing or, if repeated radiographs can not be taken, it should last at least two weeks beyond the cessation of clinical signs (Gilmore 1987). In our patient in the absence of control radiographs we advised for a 12 weeks long treatment regimen.

In conclusion, although rarely encountered in the clinical practice *Erysipelothrix rhusiopathiae* may cause discospondylitis in dogs. The prognosis seems to be favourable in the absence of mulitfocal infection like polyarthritis and endocarditis.

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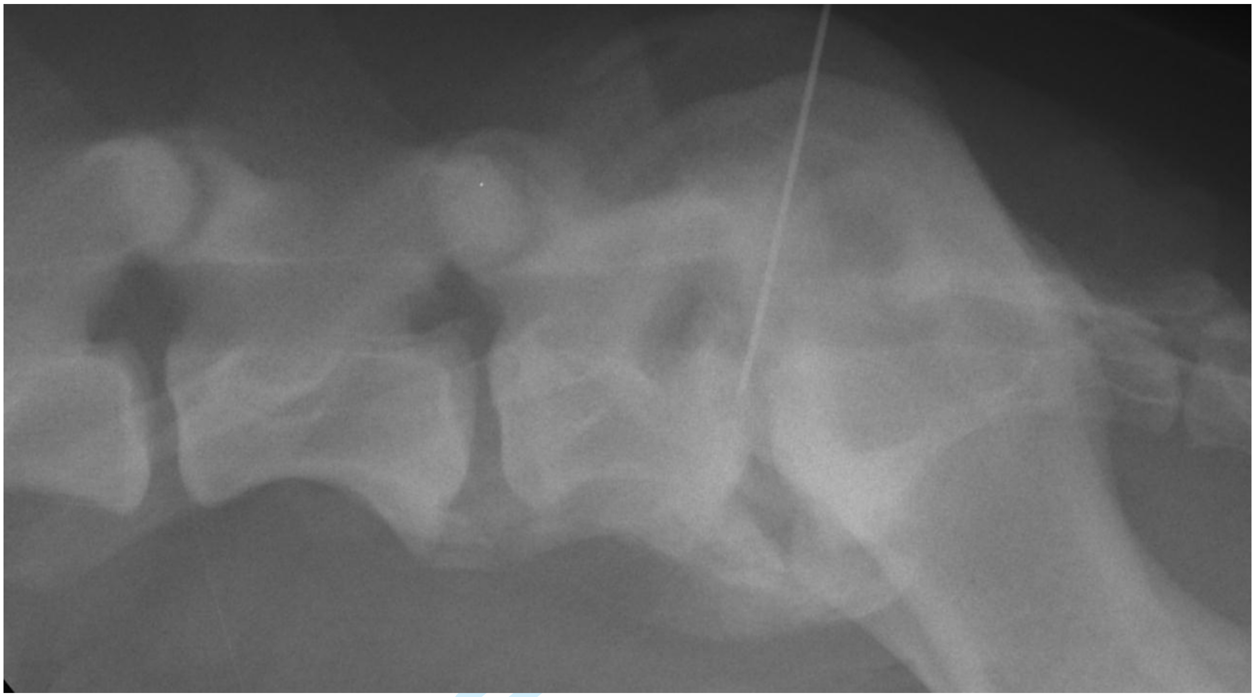
Legend of the Figures

Figure 1: Laterolateral radiograph of the lumbosacral region during the FNA aspiration. Laterolateral radiograph of the lumbosacral joint with a heavy pattern of spondylosis deformans at L6-7 and L7-S1. A collapsed LS disk space is evident plus endplate sclerosis at this disk space. The centrally located end plate lysis is difficult to visualize because of the laterally positioned superimposed spondylosis. The tip of the needle is within the center of the intervertebral disk.

Figure 2: Ventrodorsal radiograph of the lumbosacral joint. The same features seen on Fig.1 are also evident as well as the bony response of the spondylosis deformans that bridges the L7 – S1 disk space laterally. The bone lysis within the endplates is clearly identified on this view.

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